Animal Conservation

Animal Conservation. Print ISSN 1367-9430

Disease dynamics during wildlife translocations: disruptions to the host population and potential consequences for transmission in desert tortoise contact networks

C. M. Aiello[1,2], K. E. Nussear[1], A. D. Walde[3], T. C. Esque[1], P. G. Emblidge[2], P. Sah[4], S. Bansal[4,5] & P. J. Hudson[2]

1 Western Ecological Research Center, U.S. Geological Survey, Henderson, NV, USA
2 Center for Infectious Disease Dynamics, Pennsylvania State University, University Park, PA, USA
3 Walde Research & Environmental Consulting, Atascadero, CA, USA
4 Department of Biology, Georgetown University, Washington, DC, USA
5 Fogarty International Center, National Institutes of Health, Bethesda, MD, USA

Keywords
translocation; disease risk; pathogen transmission dynamics; contact networks; desert tortoise; *Mycoplasma*.

Correspondence
Christina M. Aiello, U.S. Geological Survey, Las Vegas Field Station, 160 N. Stephanie St., Henderson, NV 89074, USA
Email: caiello@usgs.gov

Editor: John Ewen

Received 2 November 2013; accepted 7 March 2014

doi:10.1111/acv.12147

Abstract

Wildlife managers consider animal translocation a means of increasing the viability of a local population. However, augmentation may disrupt existing resident disease dynamics and initiate an outbreak that would effectively offset any advantages the translocation may have achieved. This paper examines fundamental concepts of disease ecology and identifies the conditions that will increase the likelihood of a disease outbreak following translocation. We highlight the importance of susceptibility to infection, population size and population connectivity – a characteristic likely affected by translocation but not often considered in risk assessments – in estimating outbreak risk due to translocation. We then explore these features in a species of conservation concern often translocated in the presence of infectious disease, the Mojave Desert tortoise, and use data from experimental tortoise translocations to detect changes in population connectivity that may influence pathogen transmission. Preliminary analyses comparing contact networks inferred from spatial data at control and translocation plots and infection simulation results through these networks suggest increased outbreak risk following translocation due to dispersal-driven changes in contact frequency and network structure. We outline future research goals to test these concepts and aid managers in designing effective risk assessment and intervention strategies that will improve translocation success.

Introduction

Wildlife translocation has developed into a widely used tool to either reintroduce or supplement existing populations in response to the growing needs of wildlife management and conservation. The frequency and objectives of translocations worldwide are increasing in an attempt to reduce the impacts of fragmentation, habitat loss and climate change (Fischer & Lindenmayer, 2000; Thomas, 2011; Weeks *et al.*, 2011). Regardless of specific project goals, the challenge for wildlife managers is: How can wildlife translocations be executed in a manner that simultaneously minimizes risk to natural populations and to the translocated individuals?

One major risk in translocation is the threat of infectious disease to the recipient population, the translocated animals and the larger potential host community. An infectious disease is any abnormal function or change in structure of an organ or organ systems in a host due to colonization by a pathogen (any disease-causing parasite including bacteria, viruses, protozoa, fungi, helminths and ectoparasites). Unforeseen disease outbreaks can result in significant mortality or reduced fitness, and hence reduce rather than augment the population (Cunningham, 1996; Deem, Karesh & Weisman, 2001; Kock, Woodford & Rossiter, 2010; Sainsbury & Vaughan-Higgins, 2012). Many early translocations either failed or exhibited complications due to disease (reviewed in Cunningham, 1996; Kock *et al.*, 2010), necessitating the development of methods to identify and reduce disease threats (Leighton, 2002; Armstrong, Jakob-Hoff & Seal, 2003; Miller, 2007; Hartley & Gill, 2010).

During preliminary disease risk assessments, managers may attempt to prioritize pathogens based on characteristics

that signify the greatest threat to a translocation. Such ranking is often based on a pathogen's current presence or likelihood of introduction into the translocated or resident host population, the virulence or severity of disease caused in infected individuals, and anticipated transmission rates (Miller, 2007). The latter requires further knowledge of the duration and frequency of infectiousness, contact rates between infected and susceptible hosts, and host susceptibility to infection given pathogen exposure – parameters that are often unknown in wild populations and highly variable among individual hosts and environmental conditions (Anderson, 2009).

In addition to the uncertainty associated with natural disease dynamics, these assessments should consider translocation as a potentially disruptive event that may influence the parameters that define disease risk and must therefore ask: Will susceptibility to infection and disease be affected by translocation? Will the frequency of contact between hosts and, thus, pathogen exposure change? Will translocation disrupt the present structure of the population and the spatial extent of pathogen transmission? Without further investigation of potential translocation effects on key components of the host – parasite system, managers may underestimate the disease risks associated with a translocation.

Published translocations that cite post-release mortality due to disease are commonly associated with resident pathogens encountered at the release site (Ewen *et al.*, 2012). In reinforcement translocations, individuals are released into an existing population of conspecifics with a natural parasite community. When this occurs, several population characteristics may change as relocated hosts move across the landscape and interact with established residents and their parasites. Many of these changes may increase the risk of, spread, and magnitude of disease outbreaks even if all relocated animals are healthy at the time of release. In this paper, we will discuss common features observed following translocations that can affect transmission and illustrate potential consequences with preliminary data on Mojave Desert tortoises *Gopherus agassizii*. We propose future research on multi-scale processes relevant to population disease dynamics and accurate translocation risk assessments.

Dynamics of disease invasion

Disease dynamics in wild populations rely on several processes that begin with the transmission of a pathogenic parasite from an infectious host to a susceptible host, resulting in infection. This infection may or may not progress to clinical disease, which can cause symptoms that either lead to mortality or the clearance of the infection through the actions of the immune system. In some instances, the pathogen is not cleared and the infection is persistent. During infection, there are often one or more periods of infectiousness during which the host can transmit the infective stages of the parasite to other hosts or to intermediate vectors.

In epidemiological studies, the basic reproduction number, R_0, is used to quantify the transmission potential of a disease. R_0 can be defined as the number of secondary infections caused by a single infected individual introduced into a population made up entirely of susceptible individuals. In a population of N individuals with a transmission rate (β), each infective individual can, on average, give rise to βN new infections during an infectious period of $1/\gamma$, where γ is the average rate of recovery, and thus R_0 can be estimated as

$$R_0 = \beta N/\gamma$$

If R_0 is less than 1, then on average an insufficient number of hosts are infected for continued transmission and the outbreak fails to establish. Larger transmission rates (which can stem from higher contact rates or higher susceptibility to infection), long infectious periods and greater population sizes will all facilitate an outbreak. There are also some potential nonlinearities in the system that may increase the likelihood of an outbreak, particularly when there is variation in susceptibility and infectiousness between individual hosts. For example, if highly susceptible hosts are also more infectious, the likelihood of an epidemic will increase although duration of the epidemic may be shorter (Keeling *et al.*, 2002; Hudson *et al.*, 2008).

When host populations exhibit heterogeneity, mean field models may fail to capture the dynamics and network models can be used to integrate individual level variation in contact, susceptibility and transmission (Keeling & Eames, 2005; Bansal, Grenfell & Meyers, 2007). These models represent hosts as nodes in a network with connections between nodes signifying unique contacts or transmission pathways. The structure of the network influences the rate of spread and the likelihood distribution of a disease outbreak, and hence the basic reproduction number (R_0) (Cross *et al.*, 2004; Bansal *et al.*, 2007; Porphyre *et al.*, 2008). An individual's position in the network not only influences his infection risk but also his role in transmission (Fig. 1) (Christley *et al.*, 2005; Drewe, 2010). Contact networks are rarely incorporated into risk assessments, but could provide a useful tool for identifying risk at several scales. We will use these models in our pilot study to illustrate their applicability to translocation risk assessments.

Dynamics of infectious disease associated with host translocations

Stress, virulence and susceptibility

Capturing and releasing animals can result in increased stress. In addition to invasive procedures such as handling, veterinary examination, captivity and transport, translocation also contributes several subtle stressors such as an increase in population size that may intensify conspecific competition, disturbance via repeated monitoring,

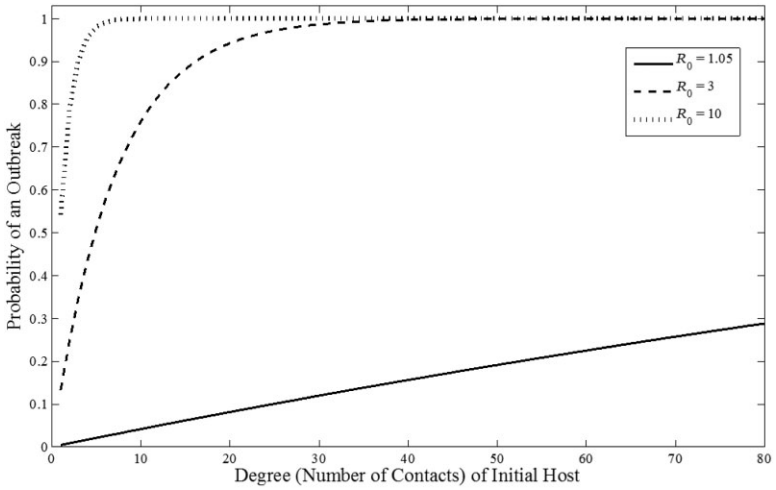

Figure 1 The probability of a disease outbreak in relation to the number of contacts made by the initial invading host with respect to the average value of the basic reproduction number R_0.

interruption of social bonds and introduction to a novel environment (see Teixeira *et al*., 2007, Dickens, Delehanty & Romero, 2010, and Parker *et al*., 2012 for a thorough review of translocation-related stress research). Repeated or prolonged exposure to multiple stressors may contribute to chronic stress where sustained physiological changes trigger immunosuppression that can increase susceptibility to infections and consequently increase pathogen virulence, both of which influence R_0 and the likelihood of an outbreak (Lafferty & Holt, 2003; Dickens *et al*., 2010).

Sudden outbreaks of disease following translocation may not involve the introduction of a novel parasite as is often assumed, but instead may result from the alteration of an existing host – parasite relationship due to stress and increased susceptibility. Stress-induced physiological changes may increase transmission rates of endemic pathogens or result in normally nonpathogenic parasites eliciting disease, which can increase disease prevalence and possibly mortality. For example, Coccidia are typically commensal microparasites in the Eurasian crane *Grus grus*, but intensity of infection can increase when host densities are high and will cause disease if immature birds are stressed (Sainsbury & Vaughan-Higgins, 2012). The behavioral and physiological changes that occur in response to acute and chronic stressors have been linked to higher rates of disease, suggesting preexisting host – parasite relationships change in the presence of stress (Dickens *et al*., 2010).

Release strategies, host density thresholds and contact rate

A common goal of translocations is to bolster population numbers and establish self-sustaining populations, which is often achieved through the release of large numbers of individuals (Griffith *et al*., 1989; Fischer & Lindenmayer, 2000; IUCN/SSC, 2013). In this way, even if some individuals succumb to mortality or disperse from the intended site, an adequate number of individuals may remain to establish and reproduce. Fischer & Lindenmayer (2000) reported that past translocations generally had higher success when more than 100 animals were released. However, recent studies show that while one-time high number releases positively effect some species, others benefit from repeated low number releases. (Shier, 2006; Linklater & Swaisgood, 2008; Faria, van Oosterhout & Cable, 2010; Shier & Swaisgood, 2012). Despite inconclusive experimental evidence, translocation guidelines recommend releasing large numbers to increase success and that multiple releases and simultaneous releases at multiple sites may have added benefit (IUCN/SSC, 2013).

Current translocation recommendations for high-number releases result in an instantaneous increase in the number of susceptible individuals effectively increasing N and so R_0, potentially fueling an epizootic that previously could not establish or that had spread through the population and died off due to an inadequate supply of susceptible hosts (Lloyd-Smith *et al*., 2005). Such a situation may be produced under natural conditions as part of an established host – parasite relationship. For example, seasonal breeding in the house finch *Carpodacus mexicanus* results in an influx of susceptible hosts, which corresponds with increased R_0 and infections by *Mycoplasma gallisepticum* (Hosseini, Dhondt & Dobson, 2004). Therefore, in populations that typically have stable numbers and low recruitment, sudden increases in host availability caused by translocation may result in significantly altered disease dynamics.

The release of large numbers may also increase contact rates between animals (Linklater & Swaisgood, 2008). High contact rates may not be uniformly distributed in the population; a few individuals have many contacts and whether these individuals are infected early in the invasion can greatly increase the likelihood of an outbreak (Fig. 1). In effect, a few highly connected hosts can be considered 'super spreaders' (Lloyd-Smith *et al*., 2005; Bansal *et al*., 2007; Hudson *et al*., 2008).

Dispersal and contact network structure

Following translocation, many species (e.g. mammals, birds, reptiles, amphibians) experience a period of rapid movement and a tendency to disperse away from the release site (Germano & Bishop, 2008; Kesler *et al.*, 2012; Le Gouar, Mihoub & Sarrazin, 2012). This response may be exploratory in nature, or tied to habitat quality, competition, social behavior, homing attempts or stress (Burns, 2005; Letty, Marchandeau & Aubineau, 2007; Dickens *et al.*, 2010; Tsoar *et al.*, 2011; Gedeon *et al.*, 2012). Exploratory or dispersing behavior varies in duration, with normal movement patterns resuming as soon as 1 day after release to as long as multiple years (Heidinger *et al.*, 2009; Nussear *et al.*, 2012). Dispersal is often viewed as a negative outcome because it can expose animals to risk of mortality from natural enemies and abiotic causes and can lead to establishment outside of the intended settlement area (Miller *et al.*, 1999; Germano & Bishop, 2008). Less appreciated is the threat dispersal presents to the population by changing disease transmission risks. Moving over large areas can result in greater overlap with conspecifics. If translocated animals have disproportionately higher contact opportunities and increase the connectivity of animals across the landscape, they could rapidly facilitate disease spread if infected. Keeling & Eames (2005) note that, 'rare long-range connections have a surprisingly large effect' on the magnitude of an infectious outbreak and highlight the importance of long-distance contacts in transmitting disease to otherwise disconnected groups (Eames, 2008).

Many animal populations have spatially clustered distributions, whether in response to a clustered resource, territoriality, or structured social or family groups (Sasaki, 1997; Grear & Schmitz, 2005; Chamaillé-Jammes, Valeix & Fritz, 2007). Meta-population structures can protect populations from epizootics, localizing outbreaks within subgroups and reducing the probability of rapid spread through the entire population (Altizer *et al.*, 2003; Lopez, Gallinot & Wade, 2005). Simulations suggest that increased connectivity between subgroups results in increased vulnerability to outbreaks, predominantly with highly infectious, low-severity diseases as high host survival will allow more time for infected individuals to move between groups (Hess, 1994, 1996; Cross *et al.*, 2004; Griffin & Nunn, 2011). Dispersal of translocated animals is likely to increase connectivity, and therefore, resident population structure and post-release movements should be incorporated into models of disease spread in risk assessments.

An example of disease risk in translocations: the Mojave Desert tortoise

Introduction to the host – parasite system

Gopherus agassizii is a long-lived, terrestrial tortoise that occurs throughout the Mojave Desert north and west of the Colorado River. The species was listed as threatened under the U.S. Endangered Species Act in 1990, largely due to declines in populations throughout their range, loss of habitat and concerns regarding an upper respiratory tract disease (USFWS, 1990, 1994, 2011). Currently, there are a number of solar energy facilities being developed across the Mojave Desert and plans for several future facilities that will result in loss of habitat and risk of direct harm to tortoises if left on site (Lovich & Ennen, 2011). Consequently, tortoises are being translocated to neighboring occupied habitat.

Disease risk assessments for this species present many challenges: information on existing diseases and how they affect populations, causative agents, transmission patterns, latency and the development of resistance is incomplete (Sandmeier *et al.*, 2009). *Mycoplasma agassizii* and *M. testudineum* are the most studied microparasites in this system and are considered the main causative agents of an upper respiratory tract disease. These pathogens are transmitted horizontally through direct contact between hosts but the potential for *Mycoplasma* spp. to survive temporarily outside the host in burrows has not been excluded (McLaughlin, 1997).

Infections with *Mycoplasma* spp. can cause recurring clinical disease interspersed by asymptomatic periods (Brown *et al.*, 1994; Christopher *et al.*, 2003; Sandmeier *et al.*, 2009). It is unclear whether *Mycoplasma* spp. infection can be cleared from host tissues or if infected desert tortoises experience lifelong infection. Captive individuals inoculated with *M. agassizii* have been recorded shedding bacteria up to 1 year post inoculation (Brown *et al.*, 1994). Prior infection does not appear to convey immunity or resistance based on studies with gopher tortoises *Gopherus polyphemus* – a host species similarly affected by *M. agassizii* infection (McLaughlin, 1997). Infected hosts appear to experience morbidity but low mortality, a condition that can still have meaningful impacts, particularly when interacting with other threats to a population (Smith, Acevedo-Whitehouse & Pedersen, 2009; Berish *et al.*, 2010; Tompkins *et al.*, 2011).

Current translocation guidelines recommend the movement of animals in good physical condition that do not exhibit moderate to severe clinical signs of disease (USFWS, 2013). This policy makes a number of assumptions: released animals with latent or mild infections will not progress to more virulent and transmissible infections, will integrate uniformly into the resident population, and will exhibit natural levels of contact and transmission. How infections in the translocated and resident population manifest and transmit through the population after release will largely depend on whether translocation alters disease parameters as discussed in above.

Current knowledge of desert tortoise translocations: implications for disease

Stress and virulence

Studies have found no statistical difference in potential stress indicators such as survival, egg production or corticosterone production (a common acute stress response

in reptiles) between translocated, resident and control tortoises (Drake *et al*., 2012; Nussear *et al*., 2012). Other stress-induced physiological changes may still weaken the immune response as observations suggest stressful environmental conditions may trigger severe upper respiratory tract disease symptoms (Sandmeier *et al*., 2009). No published studies have examined translocation effects on host susceptibility to Mycoplasma spp. infection or changes in intensity of existing infections and severity of clinical disease. Additionally, coinfections with parasites not considered in tortoise risk assessments may increase virulence of *Mycoplasma* spp. and the infectious period since naive tortoises inoculated with nasal discharge from infected individuals experienced more severe symptoms than hosts inoculated with *M. agassizii* cultures alone (Brown *et al*., 1994). If translocation affects immune response or increases virulent polymicrobial associations, rates of transmission-facilitating contact will likely increase (Anderson, 2009).

Population size and density

The number of tortoises relocated to each site during a translocation is often influenced by resident population density. Managers attempt to limit post-release densities to the average density of tortoise populations across the local recovery unit (a subdivision of the larger population) (USFWS, 2011) and encourage translocations to depopulated areas. While density limits are a positive step in preventing extreme changes in host numbers and avoiding density-dependent responses, population densities are difficult to estimate in this species (Nussear & Tracy, 2007; Inman, Nussear & Tracy, 2009). Underestimates of site resident density may result in more tortoises being released than should be, inducing a rapid change in population density. This contrasts with natural populations where increases in tortoise abundance are slow due to high juvenile mortality (Bjurlin & Bissonette, 2004) and slow recruitment (Woodbury & Hardy, 1948); as such, in undisturbed populations, host – parasite dynamics also are likely to change slowly over time.

Dispersal

Desert tortoise distributions exhibit substructuring within populations with small numbers of tortoises aggregated in discrete areas and empty habitat between (Duda, Krzysik & Meloche, 2002). While frequent interaction between adjacent groups is likely, longer distance movements greater than 3 km appear less common (Duda *et al*., 2002; Harless *et al*., 2009, 2010; Franks, Avery & Spotila, 2011). Limited movement between groups should protect tortoise populations from rapid spread of chronic upper respiratory tract disease, particularly if severe disease is triggered by low-resource conditions such as drought when tortoises responsively reduce movements across the landscape or if infection results in reduced surface activity (Brown *et al*., 1994; Duda, Krzysik & Freilich, 1999; Sandmeier *et al*., 2009). Desert tortoises frequently disperse after relocation often in the form of atypical straight-line paths, as illustrated in Fig. 2 (Field *et al*., 2007; Hinderle, 2011; Nussear *et al*., 2012). These dispersal paths can greatly exceed the maximum distances traveled by residents and are likely to connect several normally disconnected subgroups (Nussear *et al*., 2012).

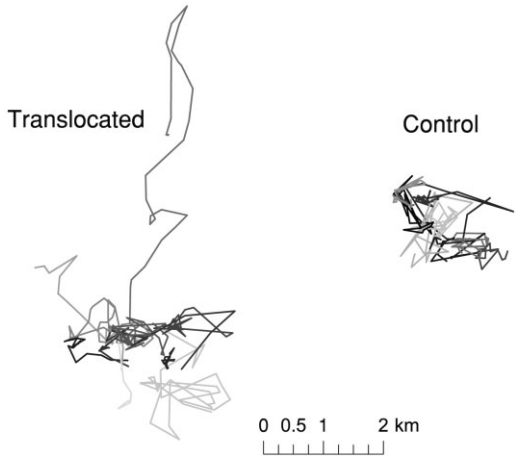

Figure 2 Movement paths of 10 control and 10 translocated tortoises during the first 2 months following release (3 April to 31 May 2008). Controls were resident tortoises at plots *c.* 2 km from the release site.

Pilot study: potential impacts to contact rates and connectivity in desert tortoises

The combined effect of increased population size and dispersal on population contact parameters can be explored with a dynamic network model. The dispersal of released tortoises creates temporal changes in spatial configuration that can influence how a pathogen travels through a population (Bansal *et al*., 2010). We used geographic locations for desert tortoises prior to and following a translocation at three translocation plots and three control plots to model dynamic contact networks and identify changes in contact rates and connectivity due to translocation. We estimated changes in disease risk by simulating infection through these networks and comparing resulting prevalence at translocation and control plots. Specifically, we asked: Did contact rate and percent animals connected in the network increase at translocation plots and not at controls immediately following translocation and, if so, how long did the perturbation last? If a network change occurred, is infection prevalence higher in residents at translocation plots than controls when we simulate infection transmission through the networks?

Materials and methods

Study site

Data for this study were collected during a translocation project that relocated animals displaced by the expansion of

Fort Irwin National Training Center, California, to nearby suitable habitat on public lands near Barstow, San Bernardino County, California, USA (Esque, Nussear & Medica, 2005). Multiple translocation sites were selected across a 1000 km^2 area based on habitat suitability, potential threats or disturbance, and existing tortoise densities (Esque *et al.*, 2005; Heaton *et al.*, 2008). Three of these sites were selected for this study because of the frequent tracking schedule of tortoises implemented at these sites and will be referred to as sites 1, 2 and 3. At each site, there are two 2.6 km^2 plots of similar habitat composition spaced at a distance of *c.* 2 km. Translocations occurred at one of the two plots at each site during 3–10 April 2008 while the other plot was left unaltered as a control.

Study animals and movement data

Surveys were conducted for samples of tortoises at all plots. Tortoises from the translocation plot are referred to as 'Residents' and tortoises at the control plots are referred to as 'Controls'. In spring of 2008, desert tortoises removed from the Fort Irwin expansion area were released within the translocation plots across the study site and following translocation, translocated (site 1 $n = 34$, site 2 $n = 33$, site 3 $n = 39$), resident (site 1 $n = 14$, site 2 $n = 20$, site 3 $n = 11$) and control (site 1 $n = 7$, site 2 $n = 14$, site 3 $n = 13$) animals were located approximately biweekly using radiotelemetry. For this study, all adult tortoises that were located within the boundary of the plot on at least one occasion in 2008 and relocated a minimum of 50 times during the post-translocation period of 3 April 2008 to 29 October 2008 were used in analysis so that animals missing for extended periods were not included.

Contact network construction and disease simulation

A contact network is typically represented with a graph of *nodes* connected by lines (referred to as *edges*) that signify a direct or inferred relationship. Each node in our graph represented a single tortoise and an edge between two tortoises signified an inferred contact. Inferred contacts were defined as a spatial proximity of 100 m or less (this distance is within the daily movement ranges observed for our tortoises) within 3 days time (all animals were typically tracked within 2–3 days time of one another). Spatial proximity or home-range overlap is often used to model networks in wild populations when interaction data are unavailable (Cross *et al.*, 2004; Godfrey *et al.*, 2010; Fenner, Godfrey & Bull, 2011). This dataset was originally collected to answer questions unrelated to social interaction and true observations of contact between tortoises were rare. We chose a fairly relaxed definition of contact to accommodate the potential error of handheld GPS (global positioning system) locations, short observation times during data collection, and temporal asynchrony of tracking events. These networks therefore reflect a nearest neighbor network and connected nodes represent tortoise pairs with a high potential for contact due to their proximity.

We constructed a dynamic contact network for each translocation and control plot that reflected the movement of animals through time. We created a pre-translocation graph for a 15-day period prior to translocation for tortoises at all plots (17 March to 31 March 2008). We then updated the graph with new edges based on changing tortoise locations at time steps of 10 days for a total of 21 time steps for the active season beginning with the first day of translocations (3 April 2008 to 29 October 2008). To simulate an infectious disease originating from the resident population, we selected a random resident to infect at time 0, and at each subsequent time step ($n = 21$) edges in the graph were updated to reflect tortoise movement, and any infected animals had a set probability of transmitting their infection to any animal they were connected to in the graph. We ran simulations with varied transmission probabilities of infected individuals to contacts from 0.1 to 1.0 in increments of 0.1 to explore the uncertainty in transmission on contact. No recovery or mortality due to infection was incorporated into simulations to reflect the characteristics of upper respiratory tract disease. Simulations were run 100 times for each transmission probability on each network. At the end of each simulation, the number of infected resident, translocated and control tortoises was recorded.

At each time step, we calculated the degree and betweenness centrality for each node (Christley *et al.*, 2005). Degree is defined as the sum of all edges connected to that node, reflecting the total number of unique individuals that tortoise may have contacted, and the degree distribution reflects the variability of hypothetical contacts across a population. Betweenness centrality is the proportion of shortest paths connecting any two nodes in the network that pass through the node of interest, signifying an animal's role as a 'bridge' between other animals. Both measures have been associated with time to infection and infection risk in both simulation and field studies of pathogen transmission (Corner, Pfeiffer & Morris, 2003; Christley *et al.*, 2005; Fenner *et al.*, 2011). Additionally, we calculated the percentage of nodes connected in the largest component of each graph. A component is a group of nodes connected to each other but to no other nodes in the network. All networks and network measures were created using the package igraph in the program R (Csardi & Nepusz, 2006; R Development Core Team, 2012).

Results

We first compared the networks for the time period immediately prior to translocation and the 10-day period including the release of tortoises, as well as the days immediately following release. Degree distributions were similar at the control and translocation plots before release, with few potential contacts between tortoises (Fig. 3a). Once translocation occurred, high degrees were frequent at translocation plots (Fig. 3b), and translocated tortoises had the most opportunities for contact. We estimated the percentage of

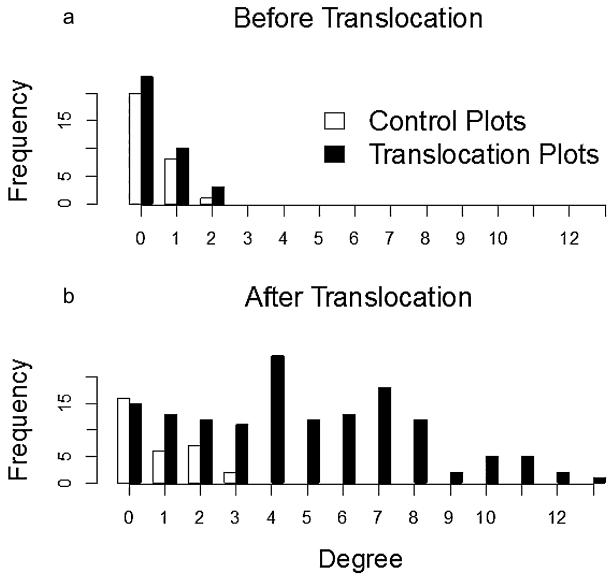

Figure 3 Degree distribution of tortoises at translocation and control plots at three sites for (a) a 15-day period prior to the release of translocated tortoises (17 March to 31 March 2008) and (b) a 10-day period when additional tortoises were released at each translocation plot over 2 days (3–12 April 2008). Degrees were based on inferred contact networks that considered two tortoises in contact based on spatial proximity < 100 m of locations made within 3 days time of each other. An individual's degree represents the total number of unique individuals a tortoise had the potential to contact.

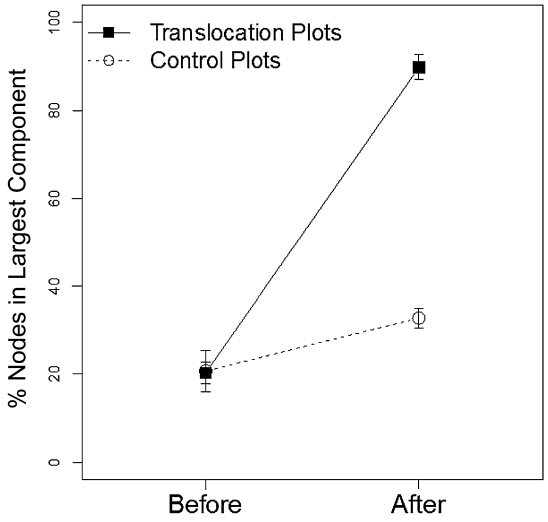

Figure 4 Average per cent of tortoises connected in the largest connected component of hypothetical contact networks for three sites prior to and following the release of animals at translocation plots. Control plots received no additional tortoises. Contact between tortoises was assumed based on spatial proximity < 100 m of locations made within 3 days time of each other. Network components are groups of tortoises connected to each other but to no other individuals in the network.

tortoises spatially 'connected' in the largest component or subgroup of each network (Fig. 4) and found that on average, 90% of tortoises at the translocation plots were potentially connected in one large subgroup compared to 33% at control plots at the time of releases.

The network changes at translocation sites were temporary, diminishing 10–20 days after translocation as released animals moved further from the site (Fig. 5). During this 20-day period following release, the translocated tortoises showed higher betweenness centrality scores compared to residents (Fig. 5). Even though local contact disruption was temporary, translocation sites often had a higher percentage of residents infected at the end of simulations than at control sites (Fig. 6).

Discussion

While wildlife managers recognize the potential for disease to affect translocation success, the potential for translocation to affect infection prevalence and the mechanisms through which this occurs needs more rigorous study. In this paper, we used the basic reproduction number R_0 and contact network methods to emphasize how changes in host susceptibility, population size, contact rates and connectivity between sub-populations can increase the likelihood of pathogen spread even if the pathogen did not originate from a translocated animal. Translocated animals, though often healthy at the time of selection, may be at high risk of acquiring infection from residents and facilitating spread. High mobility after release may increase contact opportunity, and stress associated with translocation may increase susceptibility or make even an avirulent infection more virulent. Indeed, a number of factors may increase R_0 after translocation and disrupt a potentially stable host – parasite relationship.

We describe the relevance of these translocation concerns in a heavily managed species, the desert tortoise, and show how pathogen spread may be affected by translocation using pilot data. A temporary change in spatial network characteristics occurred at translocation plots but not control plots. The change in degree distribution of our estimated translocation networks suggests higher contact rates are possible after translocation, especially for highly mobile translocated animals that were often observed within the vicinity of several unique neighbors following release. This post-release activity may also increase resident contact opportunities and facilitate increases in resident connectivity. We expect the movement of translocated tortoises away from the release site plays an important role in connecting distinct subgroups, as demonstrated by their high betweenness centrality in the first 10–20 days following release and the large percentage of tortoises incorporated in the largest connected component of networks after translocation. High degree and betweenness centrality is often associated with higher risk of acquiring and transmitting infection, especially if stress has compromised immune response (Corner *et al.*, 2003; Christley *et al.*, 2005; Fenner *et al.*, 2011; Plowright *et al.*, 2013).

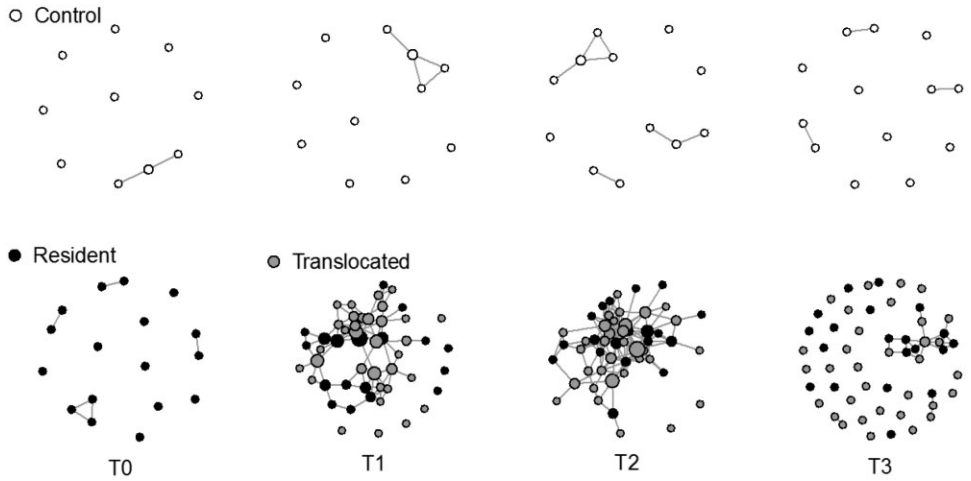

Figure 5 Inferred contact network of desert tortoises at site 2 control (top row) and translocation plot (bottom row) 15 days prior to translocation (T0; 17 March to 31 March 2008) and 10-day intervals during translocation (T1; 3–12 April 2008) and following translocation (T2 and T3; 13 April to 2 May 2008). Networks were drawn using the 'fruchterman reingold' algorithm to determine layout, therefore node (tortoise) location does not reflect geographic location (Fruchterman & Reingold, 1991). Lines connecting nodes represent an inferred contact between tortoises based on spatial proximity < 100 m of locations made within 3 days time of each other during that time step. Node size is scaled by its betweenness centrality score: a measure that reflects an individual's bridging role between other pairs or groups.

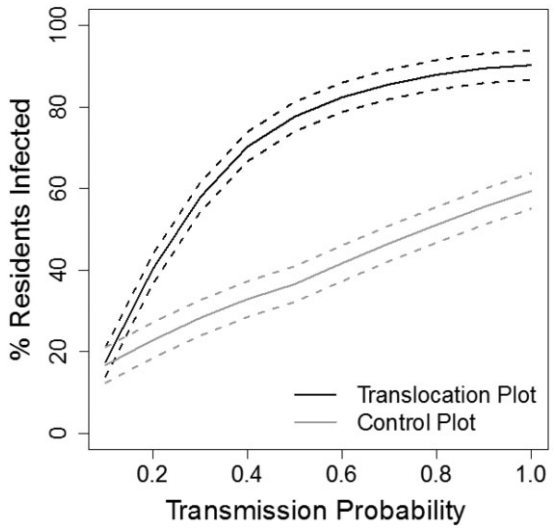

Figure 6 Infection simulation results for site 2 control and translocation plots represented by a loess smoothing curve with 95% confidence intervals. Simulations were run 100 times for each transmission probability where at each run, one random resident was infected at the start of a simulation and infection was allowed to spread based on the inferred contact networks derived from tortoise locations throughout the study period. Estimated contacts were updated at every time step to reflect tortoise movement during consecutive 10-day periods from 3 April 2008 to 29 October 2008.

Though the local connectivity changes appeared temporary, infection simulations that incorporated the movement and changing spatial configuration of these populations showed higher infection prevalence in residents at translocation networks, most notably at moderate transmission probabilities. This suggests increased disease risk at local scales, but larger impacts to population disease dynamics may also be possible. Released animals eventually moved away from the core resident group, likely returning the local connectivity to pre-translocation levels, but potentially creating connections between other groups at larger scales. Reduced clustering at the landscape level may increase outbreak risk for the wider population (Hess, 1994, 1996; Cross *et al.*, 2004; Griffin & Nunn, 2011). While this analysis suggests translocation affects contact network characteristics important to pathogen transmission, it relies on several assumptions discussed below and should be interpreted with care. This is only the first step for obtaining a more structured risk assessment for disease invasion after translocation and for developing translocation-focused research and modeling that would benefit from a risk analysis approach.

We constructed an approximation of tortoise contact networks based on radio telemetry data, but caution there are several reasons why these networks may not reflect the actual contact network. The magnitude of spatial and temporal proximity used to define a contact (100 m within 3 days), while appropriate for the coarse scale of the data available, may overestimate contacts in these populations. By estimating contact between individuals as a function of their physical proximity, we assume spatial distance between pairs is negatively correlated with contact probability, ignoring other variables that may also influence contact patterns. Translocated animals may avoid unfamiliar residents or seek out conspecifics or differences in gender between interacting animals may be important, regardless of their proximity (Serrano *et al.*, 2004; Pinter-Wollman, Isbell & Hart, 2009). We also have periods between observations when movement and contact potential is unknown.

Essentially, detailed data on true contact patterns in tortoise populations are needed to determine if such estimations are accurate.

Furthermore, even a well-documented contact network may not represent the actual *transmission* network. If long-exposure times or specific environmental conditions (e.g. high humidity in burrows) are needed to facilitate transmission between hosts, a general contact network may not be appropriate for disease transmission models. Indeed, the type, duration or location of contact can be necessary features for accurate transmission estimates as observed in both models of Sin Nombre virus prevalence in deer mice *Peromyscus* spp. and tuberculosis infections in meerkats *Suricata suricatta* (Clay *et al.*, 2009; Drewe, 2010).

There are several limitations to disease assessments in the desert tortoise system that will reflect uncertainties in the free-living, wild host – parasite systems. In addition to a lack of estimates for transmission likelihoods, the infectious period and the rate of parasite-induced host mortality, we also do not know if coinfections affect transmission, or whether infection alters host behavior such that activity and contact rates are reduced. We ran simulations using variable transmission probabilities and assumed infectiousness that lasted throughout the study period (7 months). Given the potential temporary nature of connectivity changes after translocation, a shorter or intermittent infectious period, or delayed infectiousness due to latency will influence whether short-term contact changes affect disease spread. The timing and duration of infectious period(s) will determine the time frame relevant to transmission networks; therefore, studies that provide estimates of its variation are invaluable to disease risk assessments.

Research agenda

The ultimate challenge is to collect data that we can apply to risk assessments that estimate the likelihood and consequences of an outbreak following translocation. Here, we outline a proposed research agenda on what data need to be obtained to provide a good understanding of the disease risks associated with translocation. We focus primarily on the issues relating to desert tortoise translocation, but this approach may help others in examining similar questions about host – parasite systems such as: Can we predict the transient dynamics of disease invasion with simple knowledge of infectiousness, infectious period and contact pattern? Can we predict contact networks from knowledge of population structure? Can we develop generic models that can be applied to a range of systems?

Step 1: identify the features of the parasite – host relationship at the individual level and obtain an understanding of variation between hosts

Undertake a series of transmission studies at the individual level, wherever possible, using a captive population. Estimate attributes of the infection and, in particular, understand the relationship between several features and transmission, including the likelihood of transmission with contact duration, frequency and intensity (i.e. contacts may be of similar duration but range in intensity from passive encounters, such as exploratory sniffing, to more dynamic interactions, such as combat). At the same time, seek to estimate shedding rates, the duration of infectiousness, and the influence of infection on host behavior and contact patterns.

Step 2: results from translocation and studies at the population level

Observe and quantify the effects of translocation on the contact network following experimental translocations of captive and wild animals at multiple scales. Seek to identify how the translocation influences contacts between and among translocated and resident individuals and how it disrupts previous contact patterns between residents. Proximity loggers can help record fine-resolution contact data in combination with radio telemetry data and larger scale survey data. This will help identify whether coarse scale data on distribution and abundance of residents may be used to estimate contact networks.

Step 3: application of findings to wildlife management

Combining results from steps 1 and 2 will allow managers to adjust contact networks with respect to likelihood of transmission events given levels of contact quality, predict how they may change with the proposed release, and simulate disease using parameters estimated from captive studies. If the process is successful, these models can advise managers whether a particular release site poses a high level of risk and should be avoided or whether particular release strategies may be preferred based on current population structure.

Given that epidemiology identifies the importance of heterogeneity of key host and pathogen characteristics to the transmission dynamics of populations, incorporating these features into risk assessments should be the next agenda in translocation research. Thorough knowledge of the host – pathogen relationship at an individual level will refine the parameter values applied to disease models, but equally necessary are data regarding population structure and contact rate. Most importantly, we must understand how translocation affects all of these features if our assessments are to reflect the dynamic nature of a population once hosts are added. Within the complexities of these relationships may exist dominant risk-causing characteristics that will inform managers of situations presenting the greatest impediment to translocation success.

Acknowledgements

We thank W. Boarman, A. Peter Woodman and C. Everly for collaboration and logistical support. We thank R. Averill-Murray and anonymous reviewers for helpful

comments to improve the quality of this paper. Earlier versions of this paper benefitted from review and discussions with K. Drake, R. Inman, D. Shyrock, J. Germano, A. Berger, M. Walden and F. Chen. The content of this paper was stimulated by the collaboration and discussions of members of the desert tortoise disease assessment workgroup including N. Lamberski, B. Rideout, J. Simecka, R. Swaisgood, C.R. Tracy and several others. This work was funded by the National Science Foundation Ecology of Infections Diseases grant #1216054 Invasion and Infection: Translocation and Transmission: An Experimental Study with Mycoplasma in Desert Tortoises. P.J.H. and S.B. acknowledge the support and stimulation from the Research and Policy for Infectious Disease Dynamics (RAPIDD) program of the Science and Technology Directorate, U.S. Department of Homeland Security, and the Fogarty International Center, NIH. Desert tortoise translocation research was conducted under California Fish and Game and U.S. Fish and Wildlife Service permits and funded by the U.S. Department of Defense, Fort Irwin, California. Any use of trade, product or firm names in this publication is for descriptive purposes only and does not imply endorsement by the U.S. government.

References

Altizer, A., Nunn, C.L., Thrall, P.H., Gittleman, J.L., Antonovics, J., Cunningham, A.A., Dobson, A.P., Ezenwa, V., Jones, K.E., Pedersen, A.B., Poss, M. & Pulliam, J.R.C. (2003). Social organization and parasite risk in mammals: integrating theory and empirical studies. *Ann. Rev. of Ecol. Evol. Syst.* **34**, 517–547.

Anderson, R.M. (2009). Epidemiology, In *Modern parasitology: a textbook of parasitology*, 2nd edn: 75–116. Cox, F.E.G. (Ed.). Oxford: Blackwell Publishing Ltd.

Armstrong, D., Jakob-Hoff, R. & Seal, U.S. (Eds) (2003). *Animal movements and disease risk: a workbook*: Apple Valley: IUCN/SSC Conservation Breeding Specialist Group.

Bansal, S., Grenfell, B.T. & Meyers, L.A. (2007). When individual behaviour matters: homogeneous and network models in epidemiology. *Royal Soc.* **4**, 879–891.

Bansal, S., Read, J., Pourbohloul, B. & Meyers, L.A. (2010). The dynamic nature of contact networks in infectious disease epidemiology. *J. Biol. Dynamics* **4**, 478–489.

Berish, J.E.D., Wendland, L.D., Kiltie, R.A., Garrison, E.P. & Cyndi, A.G. (2010). Effects of mycoplasmal upper respiratory tract disease on morbidity and mortality of gopher tortoises in northern and central Florida. *J. Wildlife Dis.* **46**, 695–705.

Bjurlin, C.D. & Bissonette, J.A. (2004). Survival during early life stages of the desert tortoise (*Gopherus agassizii*) in the south-central Mojave Desert. *J. Herpetol.* **38**, 527–535.

Brown, M.B., Schumacher, I.M., Klein, P.A., Harris, K., Correll, T. & Jacobson, E.R. (1994). *Mycoplasma agassizii* causes upper respiratory tract disease in the desert tortoise. *Infect. Immun.* **62**, 4580–4586.

Burns, C.E. (2005). Behavioral ecology of disturbed landscapes: the response of territorial animals to relocation. *Behav. Ecol.* **16**, 898–905.

Chamaillé-Jammes, S., Valeix, M. & Fritz, H. (2007). Managing heterogeneity in elephant distribution: interactions between elephant population density and surface-water availability. *J. Appl. Ecol.* **44**, 625–633.

Christley, R.M., Pinchbeck, G.L., Bowers, R.G., Clancy, D., French, N.P., Bennett, R. & Turner, J. (2005). Infection in social networks: using network analysis to identify high-risk individuals. *Am. J. Epidemiol.* **162**, 1024–1031.

Christopher, M.M., Berry, K.H., Henen, B.T. & Nagy, K.A. (2003). Clinical disease and laboratory abnormalities in free-ranging desert tortoises in California (1990–1995). *J. Wildlife Dis.* **39**, 35–56.

Clay, C.A., Lehmer, E.M., Previtali, A., St. Jeor, S. & Dearing, M.D. (2009). Contact heterogeneity in deer mice: implications for Sin Nombre virus transmission. *P. Roy. Soc. Lond. B. Bio.* **276**, 1305–1312.

Corner, L.A.L., Pfeiffer, D.U. & Morris, R.S. (2003). Social-network analysis of *Mycobacterium bovis* transmission among captive brushtail possums (*Trichosurus vulpecula*). *Prev. Vet. Med.* **59**, 147–167.

Cross, P.C., Lloyd-Smith, J.O., Bowers, J.A., Hay, C.T., Hofmeyr, M. & Getz, W.M. (2004). Integrating association data and disease dynamics in a social ungulate: bovine tuberculosis in African buffalo in the Kruger National Park. *Ann. Zool. Fenn.* **41**, 879–892.

Csardi, G. & Nepusz, T. (2006). The igraph software package for complex network research. *InterJournal, Complex Systems* **1695**. [accessed 10 January 2013.] (http://igraph.sf.net).

Cunningham, A.A. (1996). Disease risks of wildlife translocations. *Conserv. Biol.* **10**, 349–353.

Deem, S.L., Karesh, W.B. & Weisman, W. (2001). Putting theory into practice: wildlife health in conservation. *Conserv. Biol.* **15**, 1224–1233.

Dickens, M.J., Delehanty, D.J. & Romero, L.M. (2010). Stress: an inevitable component of animal translocation. *Biol. Conserv.* **143**, 1329–1341.

Drake, K.K., Nussear, K.E., Esque, T.C., Barber, A.M., Vittum, K.M., Medica, P.A., Tracy, C.R. & Hunter, K.W. (2012). Does translocation influence physiological stress in the desert tortoise? *Anim. Conserv.* **15**, 560–570.

Drewe, J.A. (2010). Who infects whom? Social networks and tuberculosis transmission in wild meerkats. *P. Roy. Soc. Lond. B. Bio.* **277**, 633–642.

Duda, J.J., Krzysik, A.J. & Freilich, J.E. (1999). Effects of drought on desert tortoise movement and activity. *J. Wildlife Manage.* **63**, 1181–1192.

Duda, J.J., Krzysik, A.J. & Meloche, J.M. (2002). Spatial organization of desert tortoises and their burrows at a landscape scale. *Chelonian Conserv. Biol.* **4**, 387–397.

Eames, K.T. (2008). Modeling disease spread through random and regular contacts in clustered populations. *Theor. Popul. Biol.* **73**, 104–111.

Esque, T.C., Nussear, K.E. & Medica, P.A. (2005). *Desert tortoise translocation plan for Fort Irwin's land expansion program at the US Army National Training Center (NTC) at Fort Irwin*. Prepared for US Army National Training Center, Directorate of Public Works.

Ewen, J.G., Acevedo-Whitehouse, K., Alley, M.R., Carraro, C., Sainsbury, A.W., Swinnerton, K. & Woodroffe, R. (2012). Empirical considerations of parasites and health in reintroduction, In *Reintroduction biology: integrating science and management*: 290–335. Ewen, J.G., Armstrong, D.P., Parker, K.A. & Seddon, P.J. (Eds). Oxford: Wiley-Blackwell.

Faria, P.J., van Oosterhout, C. & Cable, J. (2010). Optimal release strategies for captive-bred animals in reintroduction programs: experimental infections using the guppy as a model organism. *Biol. Conserv.* **143**, 35–41.

Fenner, A.L., Godfrey, S.S. & Bull, C.M. (2011). Using social networks to deduce whether residents or dispersers spread parasites in a lizard population. *J. Anim. Ecol.* **80**, 835–843.

Field, K.J., Tracy, C.R., Medica, P.A., Marlow, R.W. & Corn, P.S. (2007). Return to the wild: translocation as a tool in conservation of the Desert Tortoise (*Gopherus agassizii*). *Biol. Conserv.* **136**, 232–245.

Fischer, J. & Lindenmayer, D.B. (2000). An assessment of the published results of animal relocations. *Biol. Conserv.* **96**, 1–11.

Franks, B.R., Avery, H.W. & Spotila, J.R. (2011). Home range and movement of desert tortoises *Gopherus agassizii* in the Mojave Desert of California, USA. *Endanger. Species Res.* **13**, 191–201.

Fruchterman, T.M.J. & Reingold, E.M. (1991). Graph drawing by force-directed placement. *Software Pract. Exper.* **21**, 1129–1164.

Gedeon, C.I., Boross, G., Németh, A. & Altbäcker, V. (2012). Release site manipulation to favour European ground squirrel *Spermophilus citellus* translocations: translocation and habitat manipulation. *Wildlife Biol.* **18**, 97–104.

Germano, J.M. & Bishop, P.J. (2008). Suitability of amphibians and reptiles for translocation. *Conserv. Biol.* **23**, 7–15.

Godfrey, S.S., Moore, J.A., Nelson, N.J. & Bull, C.M. (2010). Social network structure and parasite infection patterns in a territorial reptile, the tuatara (*Sphenodon punctatus*). *Int. J. Parasitol.* **40**, 1575–1585.

Grear, J.S. & Schmitz, O.J. (2005). Effects of grouping behavior and predators on the spatial distribution of a forest floor arthropod. *Ecology* **86**, 960–971.

Griffin, R.H. & Nunn, C.L. (2011). Community structure and the spread of infectious disease in primate social networks. *Evol. Ecol.* **26**, 779–800.

Griffith, B., Scott, J.M., Carpenter, J.W. & Reed, C. (1989). Translocation as a species conservation tool: status and strategy. *Science* **245**, 477–480.

Harless, M.L., Walde, A.D., Delaney, D.K., Pater, L.L. & Hayes, W.K. (2009). Home range, spatial overlap, and burrow use of the desert tortoise in the West Mojave desert. *Copeia* **2009**, 378–389.

Harless, M.L., Walde, A.D., Delaney, D.K., Pater, L.L. & Hayes, W.K. (2010). Sampling considerations for improving home range estimates of desert tortoises: effects of estimator, sampling regime, and sex. *Herpetol. Conserv. Biol.* **5**, 374–387.

Hartley, M. & Gill, E. (2010). Assessment and mitigation processes for disease risks associated with wildlife management and conservation interventions. *Vet. Rec.* **166**, 487–490.

Heaton, J.S., Nussear, K.E., Esque, T.C., Inman, R.D., Davenport, F.M., Leuteritz, T.E., Medica, P.A., Strout, N.W., Burgess, P.A. & Benvenuti, L. (2008). Spatially explicit decision support for selecting translocation areas for Mojave desert tortoises. *Biodivers. Conserv.* **17**, 575–590.

Heidinger, I.M.M., Poethke, H.J., Bonte, D. & Hein, S. (2009). The effect of translocation on movement behaviour – a test of the assumptions of behavioural studies. *Behav. Process.* **82**, 12–17.

Hess, G. (1996). Disease in metapopulation models: implications for conservation. *Ecology* **77**, 1617–1632.

Hess, G.R. (1994). Conservation corridors and contagious disease: a cautionary note. *Conserv. Biol.* **8**, 256–262.

Hinderle, D. (2011). *Desert tortoises (Gopherus agassizii) and translocation: homing, behavior, habitat and shell temperature experiments*. MSc Thesis, San Diego State University, San Diego, CA.

Hosseini, P.R., Dhondt, A.A. & Dobson, A. (2004). Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. *P. Roy. Soc. Lond. B. Bio.* **271**, 2569–2577.

Hudson, P.J., Perkins, S.E., Cattadori, I.M., Ostfeld, R.S., Keesing, F. & Eviner, V.T. (2008). The emergence of wildlife disease and the application of ecology. In *Infectious disease ecology: the effects of ecosystems on disease and of disease on ecosystems*: 347–367. 11th Cary Conference, Millbrook, New York, USA, May 2005. Princeton, NJ: Princeton University Press.

Inman, R.D., Nussear, K.E. & Tracy, C. (2009). Detecting trends in desert tortoise population growth: elusive behavior inflates variance in estimates of population density. *Endangered Species Research* **10**, 295–304.

International Union for the Conservation of Nature (IUCN/SSC) (2013). *Guidelines for reintroductions and other conservation translocations, version 1.0*: Gland: IUCN Species Survival Commission, viiii + 57 pp.

Keeling, M.J. & Eames, K.T. (2005). Networks and epidemic models. *Royal Soc.* **2**, 295–307.

Keeling, M.J., Woolhouse, M.E.J., May, R.M., Davies, G. & Grenfell, B.T. (2002). Modelling vaccination strategies against foot-and-mouth disease. *Nature* **421**, 136–142.

Kesler, D.C., Cox, A.S., Albar, G., Gouni, A., Mejeur, J. & Plassé, C. (2012). Translocation of Tuamotu kingfishers, postrelease exploratory behavior, and harvest effects on the donor population. *Pac. Sci.* **66**, 467–480.

Kock, R.A., Woodford, M.H. & Rossiter, P.B. (2010). Disease risks associated with the translocation of wildlife. *Rev. Sci. Tech. OIE.* **29**, 329–350.

Lafferty, K.D. & Holt, R.D. (2003). How should environmental stress affect the population dynamics of disease? *Ecol. Lett.* **6**, 654–664.

Le Gouar, P., Mihoub, J. & Sarrazin, F. (2012). Dispersal and habitat selection: behavioral and spatial constraints for animal translocations, In *Reintroduction biology: integrating science and management*: 138–164. Ewen, J.G., Armstrong, D.P., Parker, K.A. & Seddon, P.J. (Eds). Oxford: Wiley-Blackwell.

Leighton, F.A. (2002). Health risk assessment of the translocation of wild animals. *Rev. Sci. Tech. OIE.* **21**, 187–195.

Letty, J., Marchandeau, S. & Aubineau, J. (2007). Problems encountered by individuals in animal translocations: lessons from field studies. *Ecoscience* **14**, 420–431.

Linklater, W.L. & Swaisgood, R.R. (2008). Reserve size, conspecific density, and translocation success for black rhinoceros. *J. Wildlife Manage.* **72**, 1059–1068.

Lloyd-Smith, J.O., Cross, P.C., Briggs, C.J., Daugherty, M., Getz, W.M., Latto, J., Sanchez, M.S., Smith, A.B. & Swei, A. (2005). Should we expect population thresholds for wildlife disease? *Trends Ecol. Evol.* **20**, 511–519.

Lopez, J.E., Gallinot, L.P. & Wade, M.J. (2005). Spread of parasites in metapopulations: an experimental study of the effects of host migration rate and local host population size. *Parasitology* **130**, 323–332.

Lovich, J.E. & Ennen, J.R. (2011). Wildlife conservation and solar energy development in the desert southwest, United States. *Bioscience* **61**, 982–992.

McLaughlin, G.S. (1997). *Upper respiratory tract disease in gopher tortoises, Gopherus polyphemus: pathology, immune responses, transmission, and implications for conservation and management*. PhD dissertation, University of Florida, Gainesville, FL, USA.

Miller, B., Ralls, K., Reading, R.P., Scott, J.M. & Estes, J. (1999). Biological and technical considerations of carnivore translocation: a review. *Anim. Conserv.* **2**, 59–68.

Miller, P.S. (2007). Tools and techniques for disease risk assessment in threatened wildlife conservation programmes. *Int. Zoo Yearbook* **41**, 38–51.

Nussear, K.E. & Tracy, C.R. (2007). Can modeling improve estimation of desert tortoise population densities? *Ecol. Appl.* **17**, 579–586.

Nussear, K.E., Tracy, C.R., Medica, P.A., Wilson, D.S., Marlow, R.W. & Corn, P.S. (2012). Translocation as a conservation tool for Agassiz's desert tortoises: survivorship, reproduction, and movements. *J. Wildlife Manage.* **76**, 1341–1353.

Parker, K.A., Dickens, M.J., Clarke, R.H. & Lovegrove, T.G. (2012). The theory and practice of catching, holding, moving and releasing animals, In *Reintroduction biology: integrating science and management*: 112–137. Ewen, J.G., Armstrong, D.P., Parker, K.A. & Seddon, P.J. (Eds). Oxford: Wiley-Blackwell.

Pinter-Wollman, N., Isbell, L.A. & Hart, L.A. (2009). The relationship between social behaviour and habitat familiarity in African elephants (*Loxodonta africana*). *P. Roy. Soc. Lond. B. Bio.* **276**, 1009–1014.

Plowright, R.K., Manlove, K., Cassirer, E.F., Cross, P.C., Besser, T.E. & Hudson, P.J. (2013). Use of exposure history to identify patterns of immunity to pneumonia in bighorn sheep (*Ovis canadensis*). *PLoS ONE* **8**, e61919.

Porphyre, T., Stevenson, M., Jackson, R. & McKenzie, J. (2008). Influence of contact heterogeneity on TB reproduction ratio R in a free-living brushtail possum *Trichosurus vulpecula* population. *Vet. Res.* **39**, 31.

R Development Core Team (2012). *R: a language and environment for statistical computing*: Vienna: R Foundation for Statistical Computing. [accessed 30 January 2013] (http://www.R-project.org).

Sainsbury, A.W. & Vaughan-Higgins, R.J. (2012). Analyzing disease risks associated with translocations. *Conserv. Biol.* **26**, 442–452.

Sandmeier, F.C., Tracy, C.R., DuPré, S. & Hunter, K. (2009). Upper respiratory tract disease (URTD) as a threat to desert tortoise populations: a reevaluation. *Biol. Conserv.* **142**, 1255–1268.

Sasaki, A. (1997). Clumped distribution by neighbourhood competition. *J. Theor. Biol.* **186**, 415–430.

Serrano, D., Forero, M.G., Donázar, J.A. & Tella, J.L. (2004). Dispersal and social attraction affect colony selection and dynamics of lesser kestrels. *Ecology* **85**, 3438–3447.

Shier, D.M. (2006). Effect of family support on the success of translocated black-tailed prairie dogs. *Conserv. Biol.* **20**, 1780–1790.

Shier, D.M. & Swaisgood, R.R. (2012). Fitness costs of neighborhood disruption in translocations of a solitary mammal. *Conserv. Biol.* **26**, 116–123.

Smith, K.F., Acevedo-Whitehouse, K. & Pedersen, A.B. (2009). The role of infectious diseases in biological conservation. *Anim. Conserv.* **12**, 1–12.

Teixeira, C.P., De Azevedo, C.S., Mendl, M., Cipreste, C.F. & Young, R.J. (2007). Revisiting translocation and reintroduction programmes: the importance of considering stress. *Anim. Behav.* **73**, 1–13.

Thomas, C.D. (2011). Translocation of species, climate change, and the end of trying to recreate past ecological communities. *Trends Ecol. Evol.* **26**, 216–221.

Tompkins, D.M., Dunn, A.M., Smith, M.J. & Telfer, S. (2011). Wildlife diseases: from individuals to ecosystems. *J. Anim. Ecol.* **80**, 19–38.

Tsoar, A., Nathan, R., Bartan, Y., Vyssotski, A., Dell'Omo, G. & Ulanovsky, N. (2011). Large-scale navigational map in a mammal. *P. Natl. Acad. Sci. USA.* **108**, E718–E724.

United States Fish and Wildlife Service (USFWS) (1990). Endangered and threatened wildlife and plants; determination of threatened status for the Mojave population of the desert tortoise. *Fed. Regist.* **55**, 12178–12191.

United States Fish and Wildlife Service (USFWS) (1994). *Desert tortoise (Mojave population): recovery plan*: Portland: US Fish and Wildlife Service.

United States Fish and Wildlife Service (USFWS) (2011). *Revised recovery plan for the Mojave population of the desert tortoise* (*Gopherus agassizii*): Pacific Southwest Region: US Fish and Wildlife Service.

United States Fish and Wildlife Service (USFWS) (2013). *Health assessment procedures for the Mojave Desert tortoise (Gopherus agassizii): a handbook pertinent to translocation*: Reno: Desert Tortoise Recovery Office, U.S. Fish and Wildlife Service.

Weeks, A.R., Sgro, C.M., Young, A.G., Frankham, R., Mitchell, N.J., Miller, K.A., Byrne, M., Coates, D.J., Eldridge, M.D.B., Sunnucks, P., Breed, M.F., James, E.A. & Hoffmann, A.A. (2011). Assessing the benefits and risks of translocations in changing environments: a genetic perspective. *Evol. Appl.* **4**, 709–725.

Woodbury, A.M. & Hardy, R. (1948). Studies of the desert tortoise, *Gopherus agassizii*. *Ecol. Monogr.* **18**, 145–200.